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ON ARTICULAR CARTILAGE.

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Articular Cartilage is generally looked upon as a structure which has few functions, other than those of a passive nature, to perform. Its value as a buffer for diminishing concussions, and as a medium for preventing undue friction in joints, is universally admitted. But its situation beyond the range of the vascular system, and its consequent slowness in reacting to the stimulus of injury or of surrounding disease, have led to its being regarded as a tissue which, apart from the functions conceded above, participates only passively in physiological and pathological processes, and might, save for these functions, have been without damage omitted from the system.

It seems to me that this estimate of its nature and functions has been the cause why it occupies so subordinate a place in the accounts we possess of observations on, and investigations into, diseases of joints generally; and some of the conclusions at which I have arrived from a study of normal and pathological articular cartilage are so opposed to the generally received opinions, that I venture to believe they will have some little value in modifying these opinions, and enabling us to form a more correct estimate of the forces put forth by this structure both in its physiological and diseased conditions.

It will be conceded that a knowledge of the true functions which a tissue normally possesses, is of the utmost importance in every point of view. It enables us to assign it its just value among surrounding, and, it may be, dissimilar structures, to rank it in its proper place as one of the constituent elements of the living whole, and to follow with a clear comprehension its pathological alterations *per se*, as well as their reactions on surrounding parts and on the whole organism. We cannot afford to dispense with this knowledge in regard to any portion of the body, for we see that just in proportion to our comprehension of the various normal functions, characteristics, and tendencies of organs and tissues, is the clearness of our views of

their pathological conditions. Those parts with whose normal characters we are best acquainted are those whose pathology is best understood, while we comprehend little of the pathology of such organs as the supra-renal capsules of whose physiological design we are ignorant. Therefore our advances in physiological science, whatever be their intrinsic value, are eagerly applied by the pathologist to his own researches, and always with profit and enlightenment.

The contemptible *rôle* assigned to articular cartilage has dissatisfied many. It has been remarked that, from our present basis, we are incapable of comprehending what observation teaches us of its behaviour, that we cannot, for instance, explain why cartilage is not worn away, while the hardest and most resisting structures, like the enamel of the teeth, show the effects of friction in a short space of time. In fact it is plain that, to comprehend cartilage at all, we must go back to the very beginning, and acquire a correct notion of its physiology, before we can progress beyond our present attainments in its pathology.

In the first place, and bearing in mind the universal existence of incrusting cartilage in articulations, the question naturally suggests itself,—what would occur in a joint suppose it were performing its usual functions unmutilated in every way save in being deprived of its cartilage of incrustation? Such a condition does not occur naturally under any circumstances, and experimentally it would be impossible to produce it, for the interference with other structures which would necessarily be entailed, and the known results following such interference, would vitiate or nullify any conclusions that might be sought to be drawn from the experiment. Hence we are compelled to fall back on other means of obtaining a clue to how the query should be answered; and there is but one means, and that the observation of a pathological process, which to any extent fulfils the conditions required.

In Chronic Rheumatic Arthritis (*Arthritis Deformans*) we have a disease where, while bones, ligaments and synovial membranes continue, at least in the earlier stages, to perform their functions in something approaching to the normal manner, considerable surfaces of the articular facets where bone rubs

against bone are deprived of the protective layer that incrusts them in the healthy joint. In this disease we find that, in the smaller articulations which are comparatively little exposed to pressure, the bones themselves are not perceptibly shortened, but retain nearly if not entirely their ordinary length, and, where apposed, are merely ground down to a smooth surface, the eburnation of which permits the continuance of motion to an extent and degree not very far off that they naturally enjoy. If, however, we go to the larger joints, such as the shoulder, hip, and knee, where we have in all the influence of large masses of muscle, and in some the necessity of supporting the weight of the body, intensifying the mutual pressure of the bones on each other, we find in addition a new phenomenon presenting itself. This consists in a wearing away of the bones by the pressure and attrition they are exposed to; and it goes to such an extent that the whole of the *cervix femoris* and nearly the whole of the head of the humerus are often worn away, and in time disappear in their respective joints, while similar, though less striking alterations of the same nature take place in the knee-joint. Even the eburnation (the so-called porcellanous deposit), occurring to a greater or less extent in the rubbed-down ends, is insufficient to arrest the destruction thus caused. The condition of the cartilage in Chronic Rheumatic Arthritis will be treated of further on, in the meantime it is sufficient to point out that the normal nutrition of the bone where it has lost its cartilage of incrustation is plainly insufficient to protect it against absorption from attrition and pressure.

This behaviour seems to suggest a clue to the function of articular cartilage, viz. that its purpose may be to resist the process of rubbing away continually going on. The next step is to examine how far the appearances of normal and abnormal cartilage support this suggestion.

I. Normal Articular Cartilage.

It is a striking fact that a section of healthy adult cartilage, made perpendicularly to the articular surface of a bone, reveals all the peculiarities of a structure in active growth. In its centre, somewhat nearer the joint-surface than the bone, there

is seen (Fig. 1, *b*) a series of rounded groups of cells, one to three in number, imbedded in a finely granular hyaline matrix, which forms, round each cell or group of cells, the capsule external to the cell-wall characteristic of cartilage. The cells possess diameters varying from $\frac{1}{2600}$ to $\frac{1}{1300}$ of an inch, and present clear protoplasm, and a round central nucleus of about half the diameter of the cell. The groups are arranged at tolerably regular intervals about $\frac{1}{800}$ of an inch apart, and their general shape and distribution, while indicative of active growth, give no more clue to the direction towards which they tend to multiply than do the granulation cells in any piece of ordinary granulation tissue. Beyond this *focus of central growth*, as we may call it, matters assume quite another aspect. As they approach the surface of the bone, the cells evidently pass into a state of much greater activity, preparatory to some important end they are to serve. They proliferate so actively that each group now consists of five to ten cells, or even more, and the individual cells are usually though not invariably larger, often doubling or even trebling their former diameters. And still more marked is the arrangement of the groups into rows, or something approaching to rows, arranged perpendicularly to the surface of the bone (Fig. 1, *c*). The rows are about $\frac{1}{1200}$ of an inch broad, and $\frac{1}{400}$ to $\frac{1}{500}$ of an inch long. They are not always exactly perpendicular to the bone-surface (though they are always parallel to the direction in which the surface of the bone is growing), but they form to the surface at least something approaching to a right angle, and are never parallel to it. Corresponding to this alteration in the aspect and arrangement of the cells, the hyaline matrix becomes more distinctly granular as it approaches the surface of the bone.

On comparing these phenomena with those taking place in the ossification of epiphysal cartilages, the similarity is very striking. In the latter it is easy to observe that the stages and transformations are almost identical in quality. There exists the focus of central growth (Fig. 4, *a*), consisting of roundish or irregular cells, few in number, imbedded in a homogeneous matrix, and tolerably equidistant from one another, and in this focus the arrangement of the cells indicates no particular direction of growth. As they approach the diaphysis, however, the cells multiply into groups (Fig. 4, *b*) arranged perpendicularly to the bone, forming rows or *rouleaux*

(Fig. 4, *g*) $\frac{1}{200}$ to $\frac{1}{90}$ of an inch long and $\frac{1}{400}$ to $\frac{1}{200}$ of an inch broad, and acquire at the same time a much greater magnitude than they previously possessed. That the alterations in the two structures are identical in their nature is evident from the fact that in some places appearances exactly similar may be met with in each.

The next point to be examined is the boundary line where articular cartilage passes into bone. The determination of what happens here is the central point of the whole question. It is not easy to obtain sections which show satisfactorily the changes that take place. The process I have found most satisfactory is to decalcify the bones by soaking them some weeks in dilute chromic acid acidulated with nitric acid, then wash them from the acids, make sections perpendicular to the surface of the bone by the razor or Rutherford's microtome, and tint with logwood. Sections thus obtained show the existence, between the cartilage presenting the appearances above described and the bone, of a *zone of altered cartilage* (Fig. 6, *c* to *i*) $\frac{1}{500}$ to $\frac{1}{250}$ of an inch in thickness, marked off from the rest of the cartilage by a deeply-stained border forming an undulating line. In this zone the hyaline matrix is more pellucid and takes on a different, generally fainter, tint when acted on by staining solutions, than that further removed from the bone. The cells imbedded in it reach their maximum of proliferation, and often tend to form groups more rounded (Fig. 6, *d*) or less arranged in rows than before. The margin of the bone forms a slightly uneven line (Fig. 1, *d*; Fig. 2, *g*; Fig. 6, *i*) dotted at intervals by small rounded prominences (Fig. 2, *d*; Fig. 6, *e* and *f*) projecting into the zone of altered cartilage for a distance of from $\frac{1}{300}$ to $\frac{1}{850}$ of an inch, and exhibiting at their bases a corresponding breadth. The number of the prominences bears a distinct proportion to the number of groups of cartilage-cells in their vicinity; they seem to correspond in position with them and complete as it were the harmony of their arrangement, and point besides in a direction corresponding with that of the long groups of cartilage-cells. They vary much in distinctness in different sections, but are always present, and their arrangement is suggestive of their being somehow related to the cartilage-groups. This arrangement is not fortuitous. In favourable sections it can be seen that on the group of cartilage-cells

reaching the margin of the bone, a most active proliferation and subdivision of its component cells commences at the end next the bone and quickly invades the rest of the group, which thus becomes converted into one of the prominences. In this proliferation the cells lose their capsule, and, ceasing to be recognisable as cartilage-cells, form by the process of multiplication a large mass of granulation tissue composed of small rounded cells, each about $\frac{1}{2500}$ of an inch in diameter, with granular protoplasm and large central nucleus whose diameter is about one half of that of the cell. It occasionally happens that one end of the cartilage-group still possesses its characteristic cells, while the other end is changed into granulation tissue (Fig. 2, *c*, *c*), but commonly the process once commenced is very rapid in involving the whole group. From this it is clear that the prominences are really groups of cartilage-cells which have undergone alteration. The alteration consists in a transmutation into a tissue not to be distinguished from medullary tissue such as occupies the cancelli of the spongy bones, in fact identical with it, and not unfrequently developing, like it, fat cells at various points.

It is not easy to see this process distinctly taking place. Out of a large number of sections there may be only one or two which exhibit it so clearly as in Fig. 2 of the accompanying plate, but once it has been observed in its entirety it is easy to recognize it in almost every section, and it seems to be a universal feature in the passage of the one structure into the other.

The masses of granulation tissue thus formed are next transformed into true bony tissue, but the transformation shows appearances which vary a good deal according to the activity of growth. Sometimes the masses coalesce and form a layer (Fig. 2, *e*) between the cartilage and the bone, and on the surface of the layer next the bone the cells become transformed into bone-corpuscles (Fig. 2, *f*). This is attended by a considerable increase of their intercellular substance, so that each cell separates to a little distance from its fellows, the intercellular substance becoming hard by the deposition in it of calcareous salts, and the cells taking on the outlines of bone-corpuscles with imperfectly formed and not very numerous

canaliculi. More frequently, on the other hand, the masses of granulation tissue remain separate and distinct (Fig. 3), and the peripheral cells of each mass become in the same manner transformed into layers of bone-tissue, so that a section presents islands of granulation tissue lying separated by a network of osseous substance. These variations are of interest as bearing on the pathological conditions which will fall to be considered afterwards.

Many observations have convinced me that these masses of medullary tissue, developed directly out of the cartilage cells, have at first no connection whatever with the medulla in the cancelli or with the vascular system. The observation of normal cartilage alone might be somewhat dubious, but as we shall afterwards find the process more distinctly marked in pathological states, this fact is, I believe, undeniable. I am aware that it is usual to describe prolongations of vascularized medullary tissue into the very margin of cartilage, and to assume that such prolongations are peculiar to inflammatory conditions and are the means by which cartilage is destroyed from its deeper aspect; but numberless observations made with this statement in view have convinced me of its inaccuracy, and I am satisfied that a fresh examination of the subject will be found to support in the clearest manner the statements just made as to the origin and nature of these prominences. It is true that in the insular development of bone from the prominences, examination shows that they do form an early communication with the neighbouring medulla and at the same time with the vascular system, as shown in Figure 6. The communication takes place by means of small channels (Fig. 6, *h*, *h*) eaten towards them through the already formed bone. The channels seem to be eaten by a tongue of medulla enclosing a loop or loops of blood-vessels, and which makes straight for the prominence almost as soon as it has formed. But in normal, and more especially, as will be afterwards seen, in pathological, cartilage (Fig. 3, *c*, *d*; Fig. 8, *b*, *c*, *d*) the prominences at an early stage contain no blood-vessel and have no communication with the neighbouring medulla, and yet are capable of performing the functions of medulla and developing new bone ere they have been provided with any vascular supply

or any communication with the neighbouring medullary tissue.

At the line of transformation of cartilage into bone the hyaline matrix of the cartilage disappears. The alteration in its texture which can be brought out by staining is probably a process of softening, preceding and preparing for its absorption by the influence of the cell-groups round which it lies, and of the medullary tissue into which they become transformed. It is plain that the cartilage-cells are capable of modifying the condition of the matrix around them, and that each group possesses its surrounding "territory," as Virchow calls it, over which it bears rule. The staining usually shows, even at some distance from the bone, alterations in the "cell-territories" around the groups, becoming more marked as they approach the line of transformation. The alterations are probably of the same nature as the alteration at the line of transformation, and, like it, preparatory to the absorption of the hyaline matrix.

Where epiphysal cartilage passes into bone, changes are observable parallel to those described above. At the line of transformation the long *rouleaux* of cells can be followed with great ease across the line of ossification. Precisely at this line a great change (Fig. 4, *c*) in the behaviour of the cartilage-cells becomes manifest. Short of this line the cells have assumed the appearance of long sausage-shaped rows (Fig. 4, *g*), about $\frac{1}{800}$ of an inch in length and $\frac{1}{5000}$ of an inch in breadth. The cells composing them are flattened on their adjacent sides, and the groups which they form are not unlike the aspect of the well-known *rouleaux* of red blood disks; but they present all intermediate forms between those given in the plate and those described under articular cartilage, and in some places it would be impossible to tell from the appearances whether the section had been taken from epiphysal cartilage or articular cartilage. But the moment the boundary line is passed, the cells proliferate, lose their flat form, and change into a long sausage-shaped mass of medullary tissue (Fig. 4, *d*). Between these masses the hyaline matrix dips down, in processes $\frac{1}{250}$ to $\frac{1}{140}$ of an inch in length and $\frac{1}{2500}$ to $\frac{1}{800}$ of an inch in breadth, for a short distance into the bone (Fig. 4, *e*), and is finally absorbed by the medullary masses, which pass into bone, corpuscles just as in articular cartilage, the transformation commencing at the periphery of the sausage-shaped masses (Fig 4, *f*).

It is scarcely necessary to explain that the sausage-shaped masses of medullary tissue formed out of the *rouleaux* of cartilage-cells represent here the prominences spoken of under articular cartilage. Their tips form an even line, and the conversion of cartilage-cells into medulla progresses so harmoniously in the various groups that the

line of advance is beautifully retained (Fig. 4, *c*). Some pathological conditions seem to modify this, and cause an unevenness of the line of transformation by permitting some groups to precede others in the change. These variations, however, it is foreign to our present purpose to enter into.

When the changes above described have been seen in their various stages, the observer cannot fail to be convinced that they amount to a complete demonstration of the fact that articular cartilage is continually producing new bone to supply the loss caused by the pressure sustained by the articulations. There is no other explanation possible, and, if any doubt remained, the comparison with the process of ossification occurring at the ends of the shafts by the development of the epiphysal cartilage into bone, showing as it does changes precisely similar in quality, though varying in activity, would, I submit, completely remove it. There is no doubt that cartilage is a structure admirably suited for resisting the pressure to which it is subjected. Hence the pressure is transmitted through it to the articular extremities of the bones, which are by their large size adapted to sustain and further to diffuse it. It seems probable that the spongy ends of the bones, by their size as well as by the elasticity (if the term be allowed) which their spongy structure confers upon them, succeed in reducing the pressure to its minimum and preventing its injurious action being concentrated on any one spot. They are however unable to neutralize the pressure completely, and the epiphyses would in time suffer absorption by the continual force thus acting upon them, were it not for the admirable provision of nature to replace what may be damaged. It seems probable that the spongy tissue of the articular ends of bones is being constantly renewed by a process of ossification proceeding from the articular cartilage.

Many facts in connection with diseases of joints become intelligible when they are viewed in the light of the processes described, and serve further to strengthen the conclusions arrived at; but some of these fall to be mentioned afterwards, and the others need not now be adverted to in detail.

The next point calling for attention is the condition of the cartilage where it is exposed to the attrition of the joint-movements.

Although the *pressure* of the articulating surfaces against each other seems to be transmitted through the cartilage to the bone, the *friction* to which the former is exposed cannot be similarly transmitted, but must be provided for by another arrangement. To understand this it will be necessary to return to the point whence we set out, viz. the focus of central growth. If, in examining a perpendicular section, we proceed from this point towards the joint-surface, we find that the clusters of cells lose their rounded form, and become elongated so as to lie parallel to the joint-surface (Fig. 1, *a*). As they approach it, they become flatter and flatter, and are composed of always fewer cells, so that in the immediate vicinity of the surface they are represented by isolated cells $\frac{1}{450}$ of an inch long and $\frac{1}{8000}$ of an inch broad, devoid of protoplasm and filled by a single elongated granular nucleus. Many such cells contain only a few fatty granules, and just before the surface is reached the nuclei have all disappeared, and the cells are represented by thin clefts, containing at the most a few granules of fat (Fig. 1, *a* ; Fig. 5, *c*).

These changes are plainly preparatory to the rubbing down of the cartilage which takes place on its surface, and doubtless the infinitely fine *detritus* thus produced is absorbed by the lymph-spaces opening through the synovial membrane.

By this additional observation we recognise that *cartilage, from its focus of central growth, grows towards the joint-surface as well as towards the bone,* and that it is not a structure owing its permanence merely to its elastic consistence and smooth surface. Were this all, it would speedily, like the enamel of the teeth, be worn away; but nature has provided against this by conferring on it what I have termed a focus of central growth, whence it developes centrifugally, towards the bone to replace its loss from the forces acting on it, and towards the joint to renew continually a surface which is constantly being worn away.

The last point in connection with normal articular cartilage to be considered here is its relation to the synovial membrane, a fringe of which covers its margin for a short distance, and is supplied with loops of blood-vessels penetrating to its border. If a section be made perpendicularly through these structures

(Fig. 5), the following are the changes traceable in proceeding from cartilage to membrane. Starting from the focus of central growth (Fig. 5, *a*), the groups of cells show no rapid proliferation, they occur in groups of two, and further on as isolated cells (Fig. 5, *e*) $\frac{1}{2500}$ of an inch broad and $\frac{1}{1200}$ of an inch long, while they alter in shape, becoming elongated and fusiform, their long axis parallel to the synovial membrane. They do not appear to become effete, but consist of protoplasm and nucleus seemingly unaltered save in shape. Presently the hyaline matrix exhibits a fibrous transformation (Fig. 5, *d*), becoming arranged in fibres parallel to the long axis of the cells. At the same time the cells approach more and more in outline and resemblance to those of connective tissue, the fibrous matrix becomes indistinguishable from the fibrous intercellular substance of connective tissue, and the altered structure passes gradually into the synovial fringe (Fig. 5, *b*). Such a section too, if looked at with a low power, shows that the deeper layers at least of the fringe are not superimposed on the cartilage, but are directly continuous with it, so that if the plane in which they lie were prolonged towards the middle of the cartilage, the margin of the fringe would gradually end (Fig. 5, *b* to *c*) by passing into the cartilage-layer next the joint-surface, where it is being prepared for its function of being rubbed down.

What has already been discussed seems to warrant the following conclusions:

1st. That articular cartilage is continually renewing itself from a focus of central growth, and grows in two directions.

2nd. That articular cartilage developes in the direction of the joint an effete layer suitable for being worn away by the joint movements.

3rd. That, growing also towards the bone, it fulfils the important function of reproducing the spongy ends of bones, which would otherwise be destroyed by the pressure to which they are exposed.

4th. That it fulfils this function by means of its cell-groups.

5th. That the cell-groups develope into masses of medullary tissue, which ossify at their periphery.

6th. That these are at first unconnected with the vascular system.

7th. That the synovial fringe and the articular cartilage pass insensibly into one another.

8th. That the peculiar consistence of cartilage, whereby it transmits pressure to the underlying bone, is owing to its hyaline matrix, which becomes altered and absorbed where it is no longer required.

II. Pathological Changes in Articular Cartilage.

If the preceding conclusions regarding the structure and functions of articular cartilage be correct, it may be assumed as probable that any additional changes produced by inflammation will be an intensifying of its normal processes. For in a structure shut out, like it, from the direct influence of the nervous, circulatory, and absorbent systems, their influence cannot, as in other structures, come into play in modifying and complicating the reactions due to tissue alone. Hence inflammation will show itself more in increased action and exaltation of its normal functions than in a perversion of them into a new groove. It has been stated that cartilage is a tissue insusceptible of inflammation, and observers have put themselves to great pains to refer the changes it displays in inflammation of the organs of which it forms part, to the action upon it of surrounding structures. This idea received a complete refutation at the hands of Redfern, but it has never lost altogether the hold it had acquired, and retains to the present day a prominent, though perhaps an unacknowledged place in the ideas that obtain regarding its inflammation. While freely admitting the insusceptibility of cartilage, from its very nature and position, to display its inflammation by some of the usual signs, such as redness and pain, or to participate in some of the usual results of inflammation, such as suppuration, I believe there will be no difficulty in showing that cartilage is not a passive structure in the presence of inflammation, but participates actively in it in its own peculiar way. I have as yet had no opportunities of studying the effect upon it of recent acute inflammations, such as might be sought for in Traumatic Ar-

thritis, Pyaemic Arthritis, &c., but have carefully investigated its participation in Scrofulous Arthritis (pulpy synovitis), commencing in the joint proper, and in Chronic Rheumatic Arthritis (Arthritis Deformans). In them there are invariably found active changes which cannot be attributed to anything but inflammation. A word of caution is, however, not unnecessary on this point. At the present time we are too much carried away with the idea that inflammation is inseparably linked to the presence of blood-vessels and lymph-spaces. The observations of Cohnheim and Arnold, although of immense value in correcting the beliefs that previously existed, are apt to lead us somewhat astray from a sound appreciation of inflammation as a whole. The changes in the capillaries, the existence of stigmata and stomata, and the migration of the white blood-cells through them into the lymph-spaces to form pus or, it may be, to become a part of the fixed tissues, are so attractive and striking, that we are, at the present moment, rather inclined to forget the existence of the tissues themselves and of the changes going on in them. Important as the former are, they are tending to make us in the present day underrate or even misinterpret the proliferation of cells and other tissue-changes, the knowledge of which we owe to Goodsir and Virchow, and yet in cartilage we can find only the latter, for the very existence of the former is prevented by the peculiar isolation of the tissue. It must also be remembered that prolonged inflammation of any tissue of the connective series tends to manifest itself in a perverted increase of its normal function. Chronic Periostitis, for instance, leads to increased production of new bone, Chronic Osteo-myelitis to Osteo-sclerosis, and Chronic Inflammation of a serous or synovial surface to Hypertrophy and increased secretion. The non-vascularity of cartilage must render any inflammation it may partake in also chronic, so that changes analogous to those cited can alone be looked for as evidences of its existence.

1. *Articular Cartilage in Scrofulous Arthritis* (*Tumor Albus, Fungus Articuli, Strumous Arthritis*).

The earliest changes I have been able to find in this disease are such as result in an increased development of bone. The

articular cartilage is increased in thickness, though only to a moderate extent, seldom attaining to more than double its normal measurement. This is sometimes present as a uniform thickening, sometimes in patches, in which latter case portions of it present a considerably greater depth than elsewhere, although everywhere the increased thickness is observable to some extent.

The group of cells between the focus of central growth and the surface of the bone take on a more tempestuous action, so to speak, and hurry more rapidly through their various stages. They reach a degree of development (Fig. 7, *a*) midway between those shown by normal and those shown by epiphysal cartilage. Instead of assuming, as in both of these, the form of *rouleaux* or sausages, the groups exhibit a more globular outline (Fig. 7, *a*; Fig. 3, *b*), and consist of masses of proliferating cells, sometimes possessing the most irregular shapes. Since the acids employed must considerably have modified their true shapes, I would be cautious in drawing inferences from specimens prepared in the manner already stated, but, so far as can be inferred from them, the cells seem to present a variety of forms due to their mutual pressure, and to contain large granular masses of protoplasm in their interior, with a sometimes granular, sometimes transparent nucleus. The groups still preserve their vertical direction to the bone-surface, and continue enlarging from cell-multiplication as they approach it, until they come to contain from six to twenty separate cells. The hyaline matrix (Fig. 7, *b*) shows the granular alteration far more distinctly marked than does normal cartilage, faintly indeed in a zone immediately around the groups (Fig. 7, *c*), but beyond this, densely clouded as if from a fine punctuation. It also occasionally presents fatty transformations in the form of irregularly rounded oil-drops, varying from $\frac{1}{200}$ to $\frac{1}{500}$ of an inch in diameter, with clear transparent masses in the centre of each, the size of which is from $\frac{1}{650}$ to $\frac{1}{2500}$ of an inch. Save in these respects there is little difference between the normal and the inflamed cartilage at this part.

In the zone of altered cartilage lying immediately on the bone, its elements seem in a hurry to fulfil their functions. The number of cells in each group (Fig. 8, *a*) becomes even

greater than before, and the groups differ from those of the normal structure in often passing into medullary tissue before they have arrived at the junction line (Fig. 8, *d*). It is much easier to trace the changes here than in healthy cartilage. Some of the groups show their ends next the bone converted into medullary tissue, and their further ends still unaltered (Fig. 8, *b*). Other groups are entirely changed into medulla although they are still short of the boundary line (Fig. 8, *d*). And occasionally, though rarely, a group will not only have passed into medulla, but have commenced to produce bone at its periphery (Fig. 8, *c*) some time before it arrives at the line of ossification. In such a case the bone-production, like that of medulla, commences at the end next the bone by conversion of its peripheral cells into bone-corpuscles, with increase and ossification of their intercellular substance. In this way such a group may, at its end nearest the boundary line, be encased in a capsule of newly formed osseous tissue.

The haste to develope of which these are the expressions, further shows itself in the appearance of the boundary line between cartilage and bone. Instead of preserving on the whole an unbroken level, it shows an uneven condition (Fig. 8, *g*) to a remarkable degree, being comparatively advanced in some places, and receding in others, so as quite to have lost its normal regularity. Moreover, its ossifying prominences present an unusual aspect. Instead of rounded papillæ, not higher than they are broad, they may be seen as nipple-like or even tongue-like projections into the cartilage (Fig. 8, *e*; Fig. 3, *c*), or even project so far as to assume a long irregularly club-shaped appearance (Fig. 8, *f*). The tongues of hyaline matrix still dip between them (Fig. 3, *f*; Fig. 8, *k*), but the old regularity is lost, and the ossification of the prominences, hurried as it seems to be, takes place for the most part in the insular form already described, without there being the usual amount of confluence of the medullary masses.

In the direction towards the joint the behaviour of the cartilage in Scrofulous Arthritis differs even more remarkably from the normal condition. As, passing from the focus of central growth, the cells approach nearer the surface, they assume at first their usual elongated shape (Fig. 9, *d*), and,

diminishing in size, become fusiform and contain little if anything besides the nucleus. At a distance, however, of from $\frac{1}{100}$ to $\frac{1}{150}$ of an inch from the surface, instead of passing into mere clefts, they begin to increase again in size and activity and become fusiform, subdivide so as to present two or even three cells within the fusiform capsule (Fig. 9, *c*), and give more distinct evidence of possessing an active protoplasm. The nearer they approach the surface, the more marked do these peculiarities become, and in addition the hyaline matrix is seen to become fibrous, the fibres parallel to the surface, the fibrous transformation more marked as it nears the surface, until finally the cartilage is transformed into a fibrous tissue with fusiform and oat-shaped cells, differing only from connective tissue in the firmness and cohesion of the intercellular substance.

This change is most evident, and reaches its greatest development at the margin of the cartilage, where it is bordered by the already inflamed synovial fringe, and at an early period capillary loops are found to extend from the fringe into the newly formed fibrous tissue. When it is thus provided with a vascular supply, it soon shows its sympathy with the surrounding tissues by taking on, like them, the characters of granulation tissue (Fig. 9, *b*). The process of fibrous transformation and vascularization spreads by degrees from the periphery over the entire surface, and as it thus advances towards the centre it gains at the same time the deeper peripheral layers of the cartilage, until at last there remains no cartilage at all at the periphery, but the bone is there covered by a structure which it is impossible to distinguish from granulation tissue, and whose origin from cartilage could hardly be inferred from its appearance. Thus it comes about that a steadily diminishing island of apparently unaltered cartilage often remains in the centre of the articulation, surrounded by what appears a vascularized synovial fringe, while peripheral portions, and in the knee-joint the semilunar cartilages, are already completely transformed into pulpy granulation tissue.

Such are the minute changes present in the earlier stages of Scrofulous Arthritis, and through them are produced the alterations commonly observed on the articular ends of the bones,

viz. pulpy degeneration of the cartilages, at first at the periphery, and subsequently even at the centre.

I would draw special attention to a peculiarity of this stage which is at once visible to the naked eye, but has been overlooked, and which is due to the combination of the processes described.

Of these one is continually, though at a slow rate, producing new bone, and so raising up the articular surface above its original level; the other is steadily effecting a centripetal diminution of the bone-producing cartilage. The sum of their action is that the new bone, which was at the beginning produced over the whole articular facet, soon ceases to be produced at the circumference where the belt of transformed cartilage lies. The cartilage remaining in the centre still goes on forming bone, but is constantly being destroyed at its edges by the ever-widening belt of transformation; and so it goes on till by the destruction of the last central piece of cartilage the process of bone-formation is for ever at an end. The raising up of the articular facet is, and must of necessity be, greatest in the centre, and diminishes as the periphery is approached.

This raising of the articular facet varies considerably in appearance, according to the form of the part affected by it. A flat articular surface like the head of the tibia is changed into a flat cone or pyramid. Very often the process is seen before its completion, and the head of the tibia is prolonged into a flattish truncated cone the apex of which is occupied by two smooth apparently little altered facets of cartilage, each bordered by a vascularized fringe like the normal synovial fringe, and articulating with two corresponding facets of the femur. The sides of the cone are covered with granulation tissue produced from the cartilage, and which is often studded with whitish points visible to the naked eye and not unlike tubercle, though the microscope shows that they have no such structure.

In a spherical articular surface the combined processes will result in its conversion into an egg-shaped surface. The head of the femur comes to present an ovoid elongation; and at its opposite extremity the surfaces of the condyles, which approach in form to the spherical shape, become elongated in the axis of the shaft. They may be found facetted at various parts accord-

ing to the position in which the tibia has been retained, but usually the facets are at the posterior part of their articular surfaces.

On articular facets of irregular shape the deformity produced varies according to the peculiarities of the part, but the influence of the two factors is always traceable, and is usually very obvious.

This growth of the articular facets is of course often rendered less distinct, and even reabsorbed or obliterated by the destruction of the bones in the later stages of the disease. In the earlier stages its existence favours the occurrence of the subluxations so common in Scrofulous Arthritis, and it even happens that, when subluxation occurs before the cartilage has all been transformed, the growth of the articular surfaces becomes so great as to prevent the success of any other means than an operation in restoring the bones to their proper position.

I have hitherto been unable to observe the ulceration of cartilage described by Redfern. So far as I could judge, the bone in the later stages of the disease rarely becomes exposed at any place where the cartilage still remains tolerably healthy, but generally where the alterations have substituted for the resisting cartilage a tissue unable to withstand the mutual pressure of the bones. Even at points where the pressure is strongest and the tendency to *decubitus* greatest, such as between the patella and outer condyle of the femur, I cannot say that I have been able in any case to satisfy myself as to the actual production of an erosion of cartilage from *decubitus*. On the contrary, the erosion has, in the specimens at my disposal, seemed due to a centripetal extension of the pulpy transformation inwards to the point of strongest pressure.

In Scrofulous Arthritis the changes in articular cartilage may be summed up as follows :—

1. The cartilage is increased in thickness.
2. It is transformed from the periphery to the centre into vascularized granulation tissue.
3. This is effected by a proliferation of its cells, commencing on the surface at its border, extending inwards towards the centre, and gradually involving the deeper layers.

4. Its increased activity leads to an increased production of new bone, going on as long as any cartilage remains, and therefore greatest at the centre, where it remains longest unaltered.

5. Various alterations of form of the articular facets are the result of this process.

2. *Articular Cartilage in Chronic Rheumatic Arthritis (Arthritis Deformans, Arthritis nodosa, Arthritis sicca, Malum senile Articulorum, Rheumatic Gout).*

Ever since this disease was recognised as having an existence separate from ordinary rheumatic arthritis and from gout, its causation has been a mystery. Ranked by various authors at one time with the former, at another with the latter, or again held to be a disease *sui generis*, the utmost divergence of opinion has prevailed as to its nature, and this has found expression in the names which have been bestowed upon it. But mysterious though its origin may be, it cannot be denied that such a disease exists, owing its origin probably to constitutional tendencies, hereditary or acquired, more or less allied to those which produce Rheumatism and Gout, affecting chiefly persons past middle age and leading to destruction of articular cartilage on the middle of the articular facets of bones, and to the production at their margins of large portions of bone overhanging the ends in mushroom-shaped masses, as if forced like soft mortar out from between the articulating surfaces.

The characters revealed by the microscope in the cartilages in this disease differ very materially from any yet considered. On the whole there is doubtless the same exaggeration of the normal functions as in Scrofulous Arthritis, though marked by some very striking peculiarities, but the result is a characteristic combination of growth and destruction seemingly peculiar to this disease alone.

To commence with the first onset of the disease as it may be seen on the facets of the sternum and clavicle in the sterno-clavicular articulations of a person whose other joints, phalangeal for example, show well-marked evidences of the malady. In this joint, or in others little exposed, the incipient alterations can be most readily found and most easily traced.

Before being opened such a joint seems quite normal, or the faintest feeling of knottiness of the margins of the cartilages alone betrays the pathological condition. When opened, the cartilages, articular and interarticular, have lost their translucency and are unusually yellowish, and, instead of having smooth polished surfaces, are rough and even granular. A section reveals the joint-surface of the articular cartilage proliferating and forming actively growing rounded cells, with fibrillation of the hyaline matrix, identical in nature with the changes described under Scrofulous Arthritis and depicted in Figure 9.

Between the focus of central growth and the bone the hyaline matrix is sometimes fibrous, sometimes, especially in less aged subjects, more granular than usual, the granular alteration most marked in the middle of the interspaces between the cell-groups. The cell-groups proliferate more abundantly than normally, but *show a tendency to pass more into groups of two or three or even into single cells than into the large aggregations* forming the groups in Scrofulous Arthritis. The zone of altered hyaline matrix next the bone seems to be absent. The line of ossification is quite irregular, just as in Scrofulous Arthritis, and the islands of medullary substance produced by the transformed cartilage cell-groups appear, as in it, at the margin of ossification or even beyond it. The islands of medullary substance are more sparing in number, and the newly-formed bone between them seems to be unusually dense and disproportionally abundant. This is caused by the *transformation of the single cartilage-cells or groups of two or three cells directly into bone-corpuscles without having passed through the intermediate stage of medullary tissue.*

This forms a predominant feature in all stages of the disease under consideration, and is most distinctly to be seen in a joint where the malady has passed the incipient stage and attained full development. Let us take, for example, a phalangeal joint where the external knotting shows the fully-developed disease. On opening it, the articular ends are found grooved from front to back with deep ruts or grooves often nearly a quarter of an inch in depth, each groove corresponding to a projecting ridge on the opposing facet. The margins of the cartilages are pro-

longed into bony masses, not projecting into the joint nor into the normal tissues external to it, but forming as it were an enlarged base (Fig. 10, *b*, *h*), with which are connected the thickened ligaments and synovial capsule. The ridges running antero-posteriorly on the articular ends, are seen on section to be composed of dense bone, arranged in Haversian systems with vessels in their interior, forming a texture nearly as compact and little vascular as the compact tissue of the diaphyses of the long bones. The dense bone seen in these ridges by its hardness produces, owing to the movements of the articulation, the corresponding grooves in the opposed surfaces, and patches of still surviving cartilage are found here and there on the ridges and in the grooves.

The mode of formation of the compact bone may be seen by examining thin sections through any of the patches of cartilage which remain. The superficial layers of the cartilage will be absent as if scraped away (Fig. 11, *a*), but the remaining deeper part shows the cell-groups (Fig. 11, *c*, *d*) in active proliferation, tending however to form groups of two or three cells, or even scattered single cells. The single cells are most numerous as they approach the surface of the bone, and there they become transformed into bone-corpuscles, thus increasing the circumference of the Haversian systems by contributing additional layers to it.

The details of their conversion are: at the immediate margin of the bone such a thing as a group of cells is a rarity, they have broken up into isolated round cells, unusually transparent. When one of these comes so near the bone as to be in contact with it (Fig. 11, *e*), it becomes surrounded with a halo of ossified matter, commencing at the point of contact, running rapidly round the cell, and forming a ring round it $\frac{1}{5000}$ to $\frac{1}{2500}$ of an inch in breadth. It looks as if this zone of ossification were formed by deposition of calcareous salts in the hyaline matrix, but close investigation of this point shows it to proceed rather from an ossification of the capsule and wall of the cell spreading outwards and causing absorption of the matrix. The cell itself is now soldered on to the convex periphery of the Haversian system and forms a slight rounded prominence upon it. The same process occurring in other cells soon causes the appearance

of prominence at the part to be lost, and the cell passes gradually through the forms intermediate between the cartilage-cell and the bone-corpuscle, till it finally becomes similar to the other bone-corpuscles, with a distinct division into protoplasm and nucleus.

Along the growing margin of the bone the newly-formed bone-cells are large and rounded (Fig. 11, *h*), and to some extent are crowded into clusters corresponding with the points of most active cartilage-growth; but, as they come to occupy situations more remote from the margin and nearer the centre of the Haversian system, they diminish in size (Fig. 11, *f*), and the distinction between nucleus and protoplasm becomes less marked.

There are thus formed at the margin of ossification a series of Haversian systems (Fig. 11), each arranged in concentric layers around its governing vessel, appearing to project like rounded masses into the cartilage. The line of ossification (Fig. 11) thus becomes irregular, the irregularity being due as it were to a series of segments of bony circles projecting into the cartilage. In time the masses of compact bone, favoured by the joint movements and the alteration of the surface of the cartilage, appear through the cartilage, and, scraping at every movement on the parts opposite them, plough furrows in it, and these furrows and ridges are the more distinct, the more nearly the joint is a true ginglymus.

In time the cartilage is thus all scraped off from the middle of the articular facets, and then the bone meets bone. The friction which now ensues is too much for the bone to resist, deprived as it is of any cartilage which might have replaced the rubbed down portions, and accordingly the compact bone and its Haversian systems are rubbed away (Fig. 10, *a*), just as one stone might be rubbed down by another, the resulting facet cutting off all projecting parts irrespective of their central vessel, the vascular canals themselves being even laid open (Fig. 10, *f*). Hence are formed the hard facets called "porcellanous deposit," being simply compact bone without any special deposit. In large joints, where weight and pressure must be borne in addition to the friction, the work of destruction goes on with irresistible force, till considerable portions of

the bones have disappeared, and nothing puts a stop to the process unless ankylosis occur, a termination which, in joints favourably situated, is occasionally met with.

At the periphery of the cartilage the process is identical in its nature though differing in its results. A section shows that the new bone formed at the peripheral part of the flat portion of the facet is both ground away and pressed outwards so as to form a flat collar (Fig. 10, *b*) within the attachment of the ligaments. The cartilage at the extreme outer margin, where the articular facet passes into the side of the bone (Fig. 10, *g*), and which is in intimate relation to the ligaments, is able to proliferate unchecked. It becomes altered so as to assume almost the characters of epiphysal cartilage. By rapid proliferation it comes to form a bulky outgrowth from the bone (Fig. 10, *g*). Its hyaline matrix (Fig. 12, *a*) appears less pellucid and more granular and striated, the striation for the most part perpendicular to the surface of the bone. Its cell-groups (Fig. 12, *b*) are changed into longitudinal rows, somewhat as in epiphysal cartilage, but more slender, composed of smaller and fewer cells; these rows lie parallel to the fibrillation and at right angles to the bone. As they approach it, they tend to break up into single cells, so that by the time the bone is reached they exist no longer as groups but as solitary cells. These are, in their turn, on reaching the margin of ossification, transformed into bone-corpuscles (Fig. 12, *g*) by the process already described, without having undergone a transmutation into medulla. From a continuance of the process arise the fungous masses of bone, forming so marked a feature of the disease as to have gained for it the name of Arthritis Nodosa.

To sum up:—

(1) The cartilages proliferate in the same manner as in Scrofulous Arthritis, but with some distinguishing peculiarities, viz.:

(2) The cartilage-cells pass into bone-cells without having become medullary tissue.

(3) They form, by means of Haversian systems, a dense layer of compact bone, which, after the destruction of the cartilage, is worn into facets.

(4) The margins of the cartilage, similarly affected, form osseous masses overhanging the ends of the bones.

In conclusion, the facts briefly detailed above, seem to render necessary a revision of the present doctrines concerning many of the deformities and diseases connected with joints, for it cannot but be that they are considerably influenced by the process of new bone-formation, which has been seen to be the main function of articular cartilage.

It must be admitted that we at present understand little of the forces at work in modifying the forms and dimensions of articulations. In undertaking the cure of a congenital club-foot, for example, we know that we have to deal with bones still possessing their fœtal forms and differing greatly, both in size and shape, from those which are present in a well-formed limb. By properly directed force we succeed in giving the bones a shape not far removed from that which they ought to have had. It is evident that a power spontaneously to mould themselves must reside in the bones, and hitherto the periosteum and medulla have been the only structures in a fully-formed bone that have been shown to possess this function. But if the functions claimed above be admitted as proven, it will be necessary to concede to articular cartilage a power of influencing the length of the bones similar to that possessed by periosteum, of influencing their breadth. In the changes produced in the form of the tarsal bones during the cure of club-foot, therefore, it will in future be requisite to recognize the importance of the office performed by the joint-cartilages in supplementing the surgeon's efforts to restore the bones to their proper form and size.

Doubtless also the disease of the knee-joint known as "knock-knee" (genu valgum), owes its causation less to a subacute inflammatory condition of the bone as hitherto assumed, than to some alteration leading to an interference with the peculiar function of articular cartilage. I have as yet had no opportunity of examining such a case, or any case of similar deformity occurring in other articulations.

But it would be tedious to dwell upon the application, to almost every joint-affection in which cartilage is, however

remotely, implicated, of an attempt, of which the preceding is a condensed *résumé*, to fill up what has long been a serious gap in our knowledge of the physiology and pathology of the articular surfaces of bones.

EXPLANATION OF PLATES.

Plate I.

Figure 1. Perpendicular section of the articular cartilage on the upper surface of a healthy adult astragalus. *a.* Cartilage developing towards the joint into a non-cellular structure suitable for being destroyed by the friction movements of the articulation. The cells are replaced by mere clefts in the hyaline matrix; *b.* Focus of central growth whence the cartilage grows in all directions; *c.* Cartilage destined to form new bone, and growing towards the bone in elongated groups and rows of cells; *d.* Level surface of the bone marked by slight prominences, which, in the drawing, are not shown so distinctly as they should have been. Magnified 150 diameters.

Figure 2. Section through the boundary line between bone and articular cartilage from the upper surface of the tibia, in a slightly inflamed knee-joint (scrofulous synovitis). *a.* Granular hyaline matrix of cartilage; *b.* Elongated groups of proliferating cartilage-cells destined to form new bone; *c.* The same undergoing by proliferation a transformation into medulla; *d.* Prominences of medulla confluent with each other at their bases *e*; *f.* Development of the deeper surfaces of the prominences into bone; *g.* Wavy boundary line between articular cartilage and bone; *h.* Prolongations of the hyaline matrix of cartilage undergoing absorption. Magnified 320 diameters.

Plate II.

Figure 3. Section through the boundary line between bone and articular cartilage of the tibia in a case of scrofulous arthritis of the knee-joint. *a.* Granular hyaline matrix; *b.* Proliferating groups of cartilage-cells; *c.* Group of cartilage-cells transformed into medulla, and developing at the end next the bone into a cup of osseous tissue; *d.* Groups of cartilage-cells transformed into medulla, and passing at the periphery into osseous tissue; *e.* Osseous tissue forming an irregular boundary line between bone and cartilage; *f.* Prolongation of the hyaline matrix undergoing absorption; *g.* Fat-cells. Magnified 320 diameters.

Figure 4. Section through boundary line between diaphysis and epiphysal cartilage from the tibia of a boy of ten. *a.* Zone of central growth; *b.* Proliferation of the cartilage preparatory to the formation of bone; *c.* Boundary line between cartilage and bone; *d.* Sausage-shaped masses of medulla formed out of proliferating cartilage groups; *e.* Processes of hyaline matrix undergoing absorption; *f.* Bone developed by the periphery of the sausage-shaped masses of medulla; *g.* *Rouleaux* of cartilage-cells before their transformation into medulla; *h.* Portion of the cartilage next the epiphysis. Magnified 150 diameters.

Plate III.

Figure 5. Perpendicular section through the joint-surface of healthy articular cartilage at its junction with the synovial fringe. *a.* Zone of central growth; *b.* Synovial fringe passing gradually into cartilage; *c.* Effete superficial layer of cartilage continuous with synovial fringe; *d.* Fibrillation of the hyaline matrix where the cartilage is passing into synovial

fringe; *e.* Transformation of cartilage-cells into the oat-shaped cells of the synovial fringe. Magnified 200 diameters.

Figure 6. Section through articular cartilage and bone from the lower end of a femur expanded by a vascular tumour. *a.* Hyaline matrix of the cartilage; *b.* Proliferating cartilage cell-groups; *c.* Boundary line between normal and altered cartilage; *d.* Proliferating cartilage cell-groups; *e.* Cartilage cell-groups transformed into medulla; *f.* The same becoming transformed at the periphery into bone; *g.* Bone; *h.* Canals eaten through bone by a tongue of vascularized medulla going to the transformed cartilage cell-groups; *i.* Border-line between bone and cartilage; *c* to *i.* Zone of altered cartilage. Magnified 200 diameters.

Plate IV.

Figure 7. Articular cartilage in Scrofulous Arthritis. *a.* Proliferating cartilage cell-groups. *b.* Granular hyaline matrix. *c.* Clear zone of hyaline matrix around the cell-groups. Magnified 500 diameters.

Figure 8. Section through articular cartilage and bone in Scrofulous Arthritis. From the head of the tibia. *a.* Proliferating cartilage cell-groups; *b.* The same transformed into medulla at the ends nearest the bone; *c.* The same completely transformed into medulla, and developing into osseous tissue at their ends nearest the bone; *d.* Cartilage cell-group transformed into medulla; *e.* Cartilage cell-groups transformed into medulla and producing osseous tissue at the boundary line: that to the left has become connected with the vascular system; *f.* The same, club-shaped; *g.* Irregular boundary line; *h.* Fat-cells; *i.* Bone; *k.* Hyaline matrix. Magnified 150 diameters.

Plate V.

Figure 9. Section of the joint-surface of articular cartilage in Scrofulous Arthritis. From the upper end of the tibia. *a.* Joint surface; *b.* Cartilage converted into granulation tissue; *c.* Isolated cartilage-cells developing into rounded cells; *d.* Isolated elongated cells. Magnified 300 diameters.

Figure 10. Section through the margin of the articular facet of a phalanx in Chronic Rheumatic Arthritis. *a.* Articular facet formed of rubbed-down compact bone (porcellanous deposit); *b.* Newly-formed compact bone pressed outwards; *c.* Haversian canals; *d.* Medulla; *e.* Haversian systems; *f.* Haversian canals laid open by attrition; *g.* Proliferating articular cartilage developing into *h*; *h.* Compact bone; *k.* Articular cartilage developing into bone *b*. Magnified 100 diameters.

Plate VI.

Figure 11. Section through a patch of articular cartilage on a "porcellanous" facet in Chronic Rheumatic Arthritis. *a.* Joint-surface. The superficial layers have been scraped away by the joint movements; *b.* Granular hyaline matrix; *c.* Proliferating cell-groups; *d.* Isolated cartilage-cells; *e.* The same soldered to the bone by a calcareous zone; *f.* Haversian systems; *g.* Haversian canals; *h.* Clusters of bone-corpuscles formed out of cartilage-cells. Magnified 400 diameters.

Figure 12. Section through the boundary line between *g* and *h* in Figure 10, Plate V. *a.* Fibrous transformation of hyaline matrix; *b.* Cartilage cell-groups; *c.* Bone; *d.* Medulla; *e.* Process of vascularized medulla, incipient Haversian canal; *f.* Fat-cells; *g.* Isolated cartilage-cells soldered to the bone and becoming transformed into bone-cells; *h.* Groups of bone-corpuscles formed out of cartilage-cells; *i.* An isolated cartilage-cell. Magnified 400 diameters.

Fig. 1.

Fig. 2.

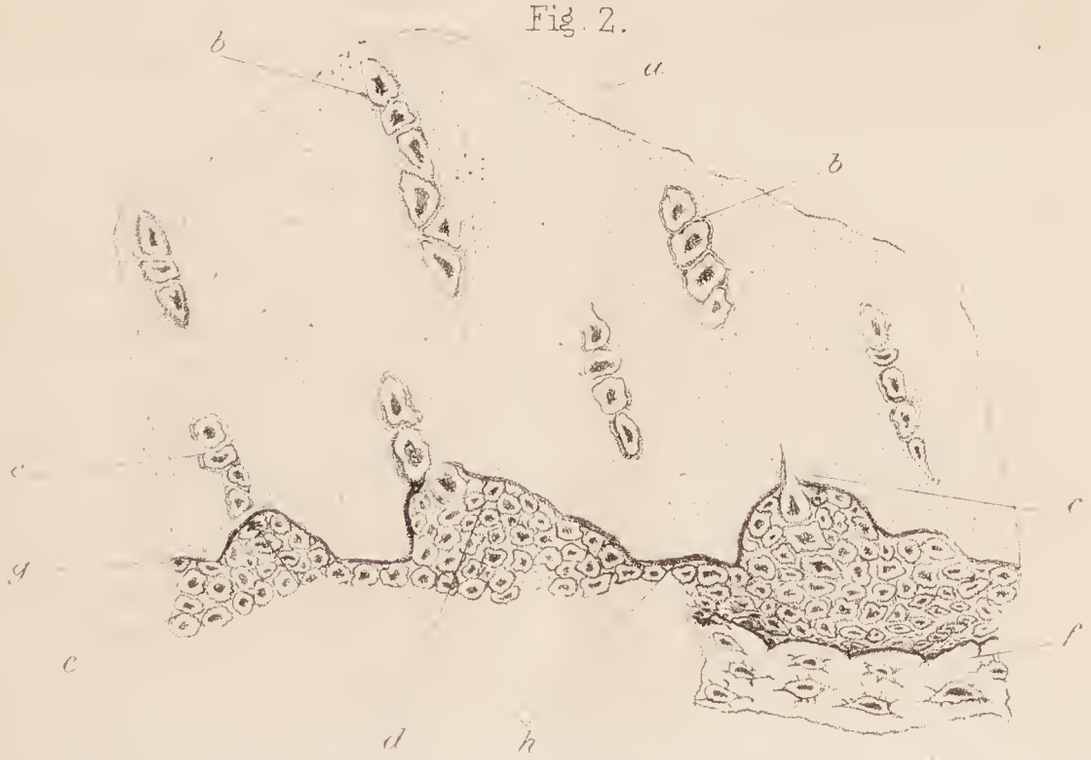


Fig. 3.

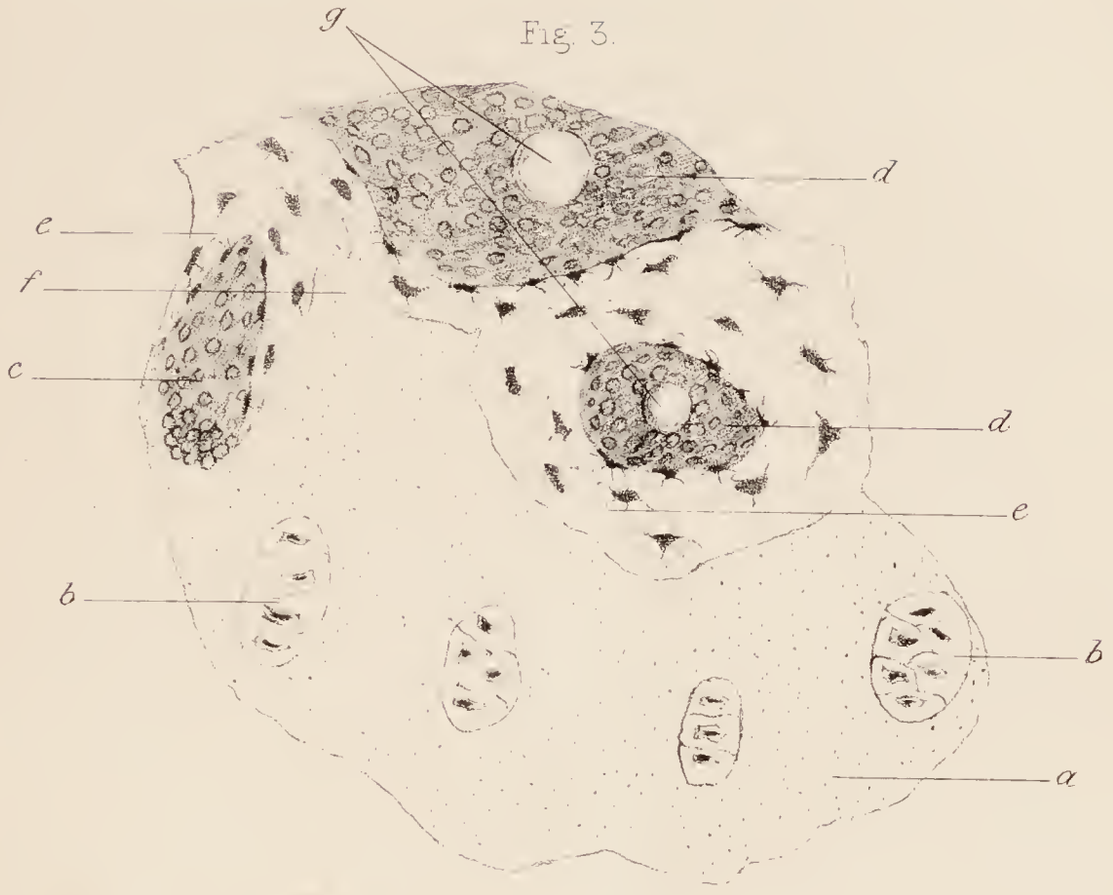
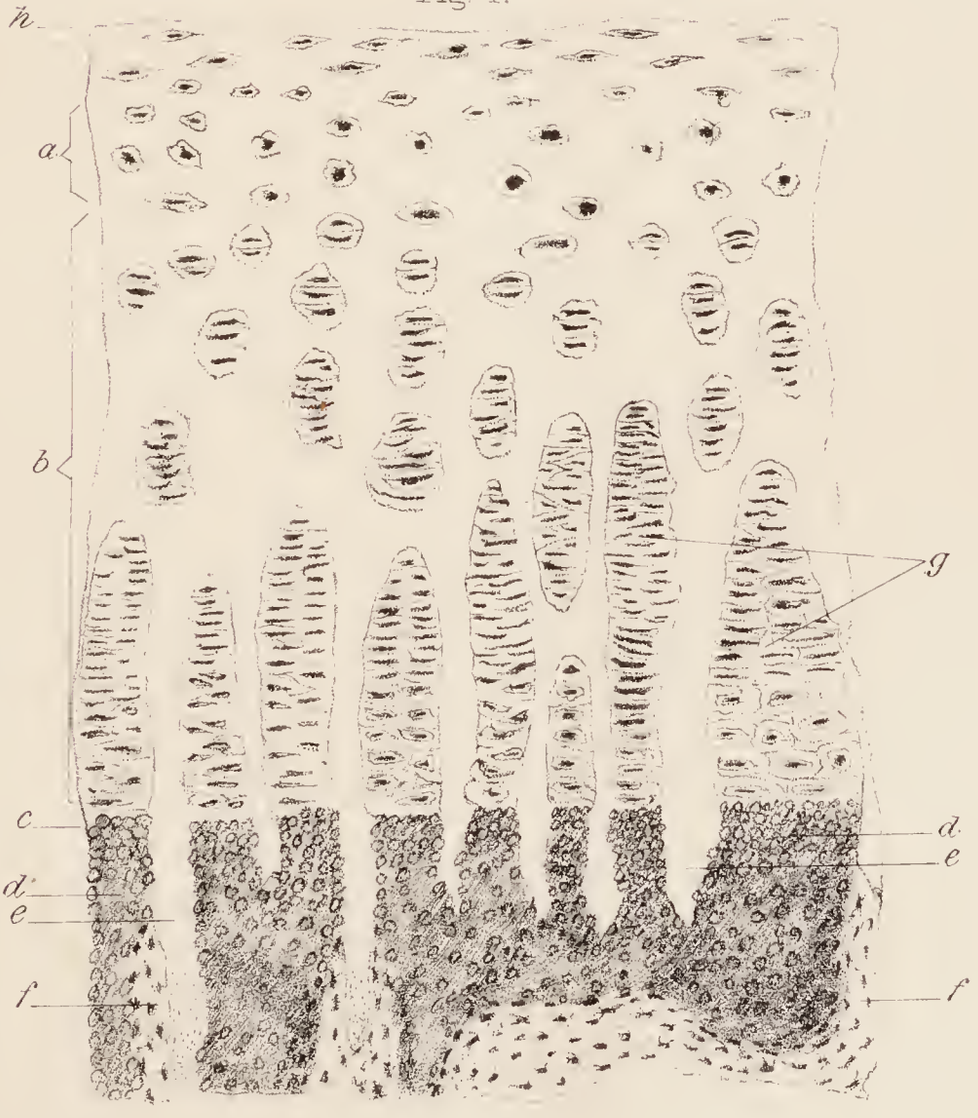


Fig. 4.



ARTICULAR CARTILAGE.

Fig. 5.

Fig. 6.

ARTICULAR CARTILAGE.

Fig. 7.

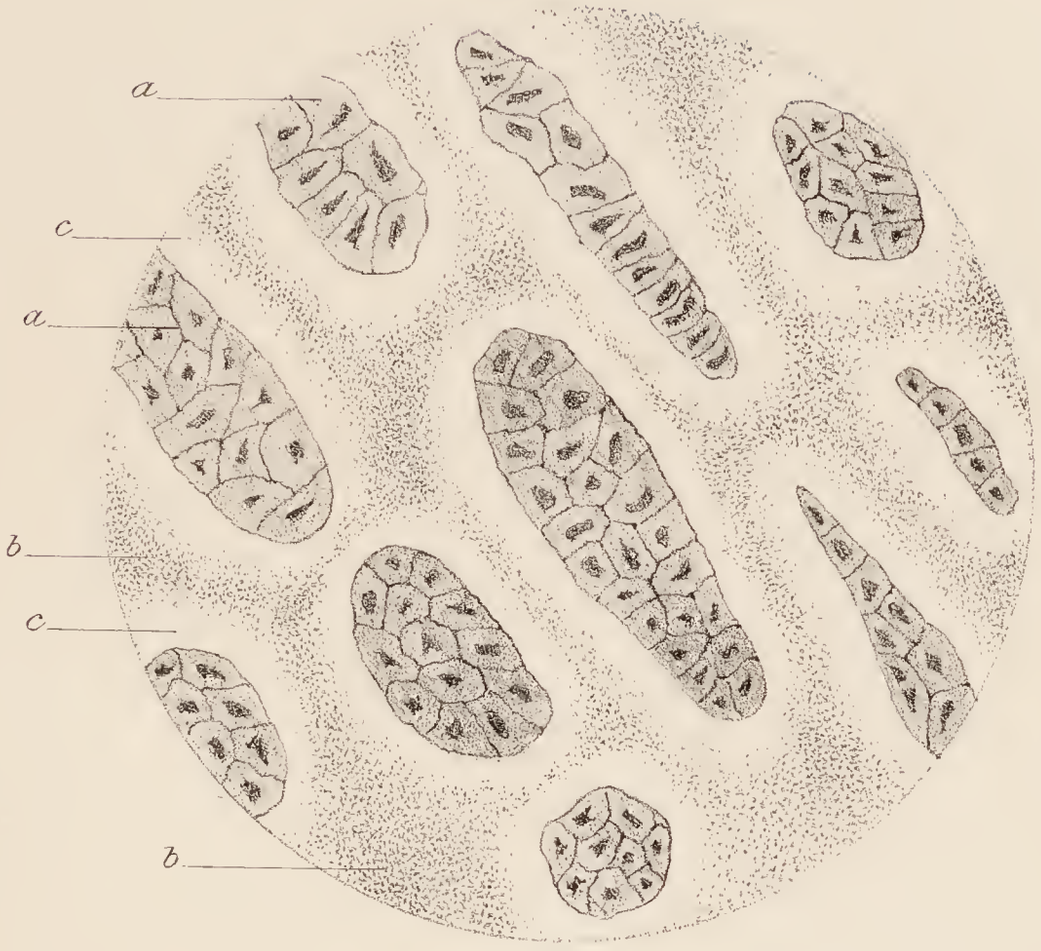
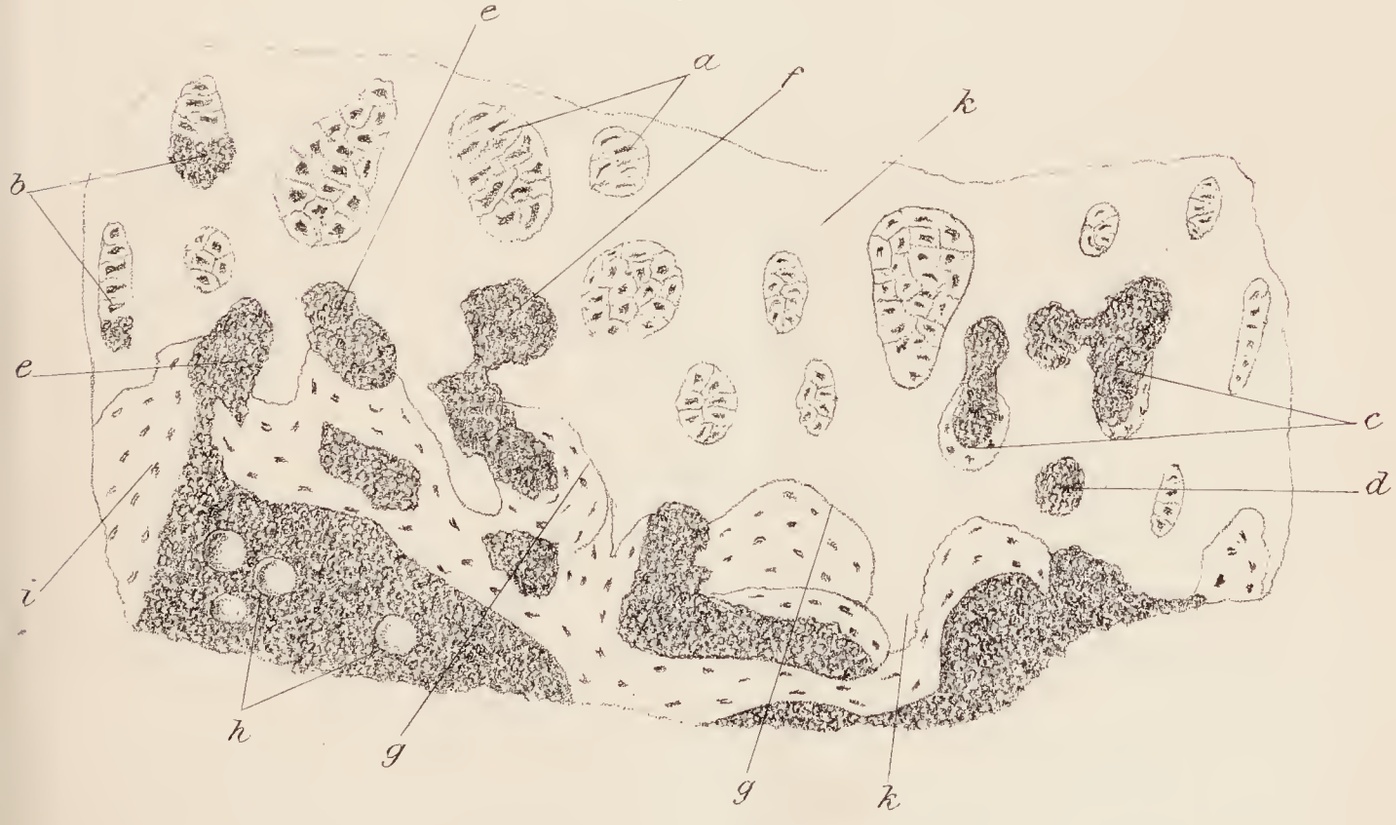


Fig. 8.



ARTICULAR CARTILAGE.

Fig. 9.

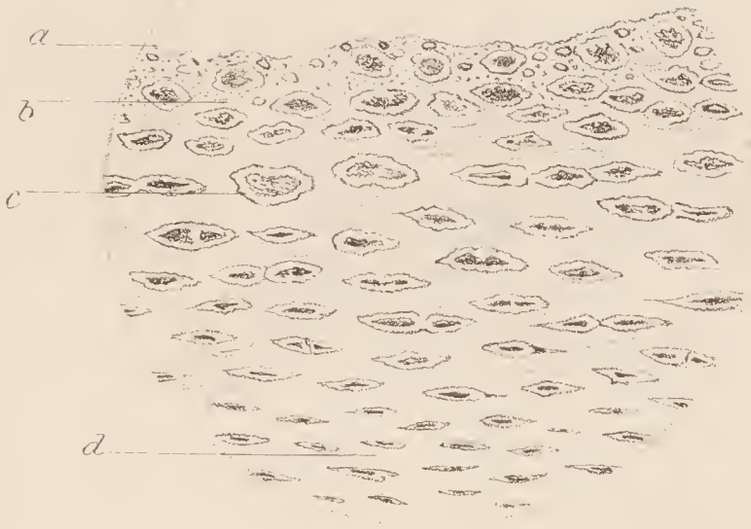


Fig. 10.

ARTICULAR CARTILAGE.

Fig. 11.

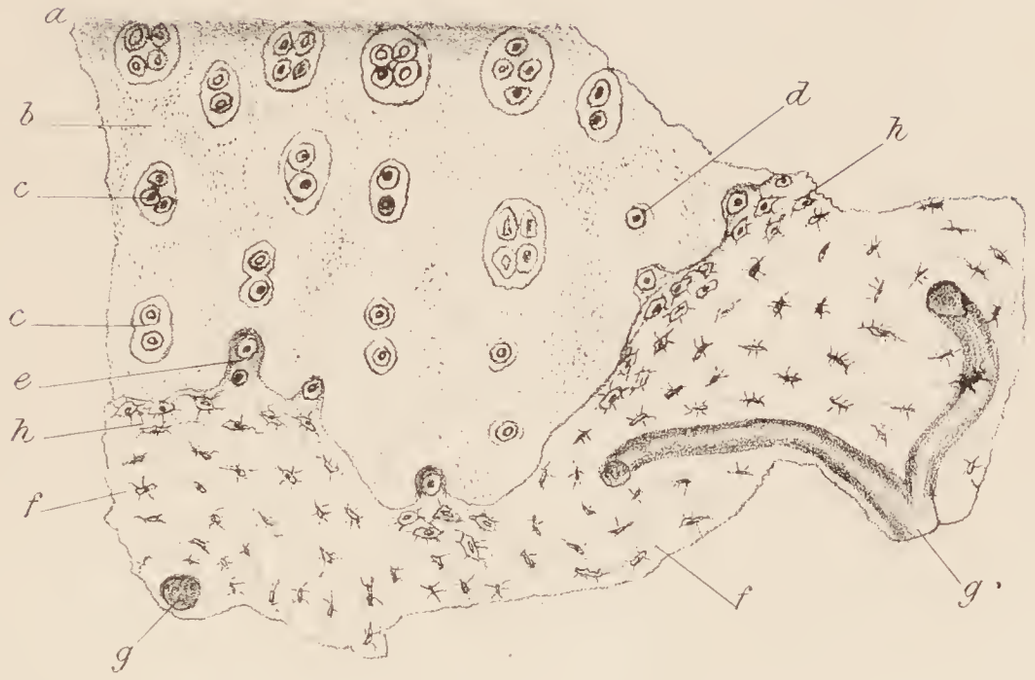
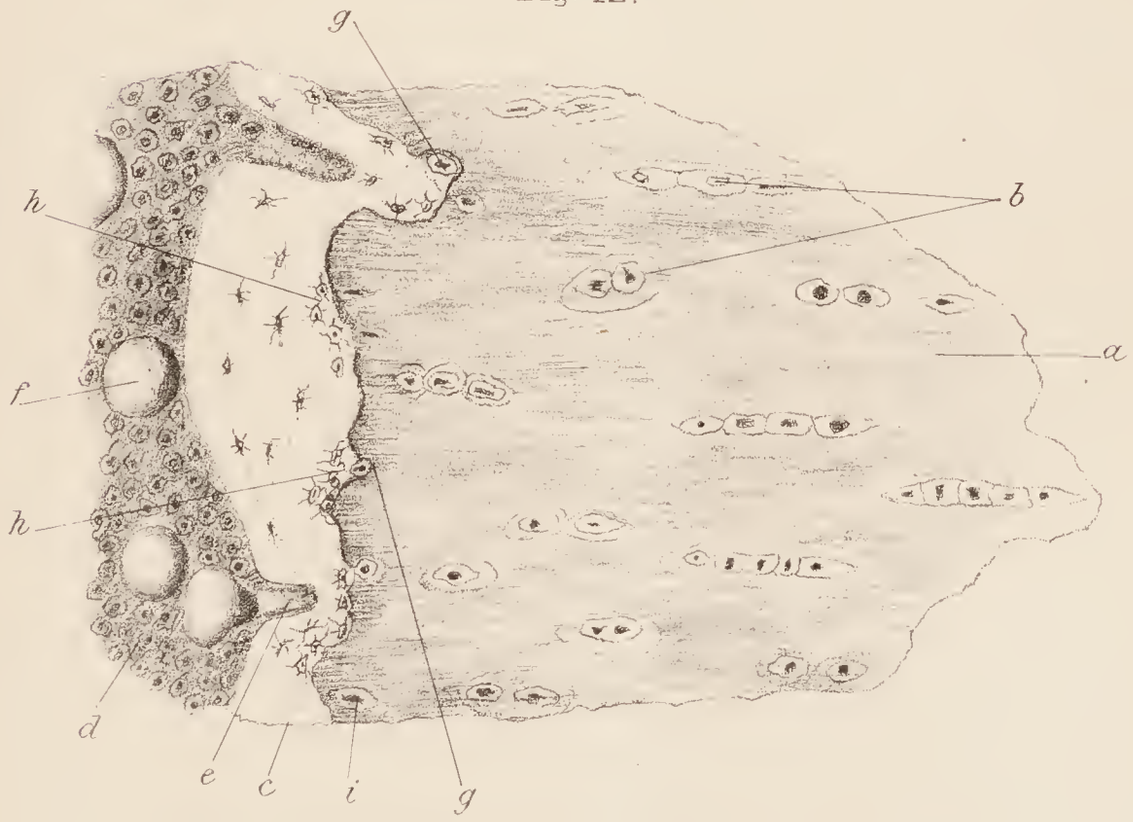


Fig. 12.



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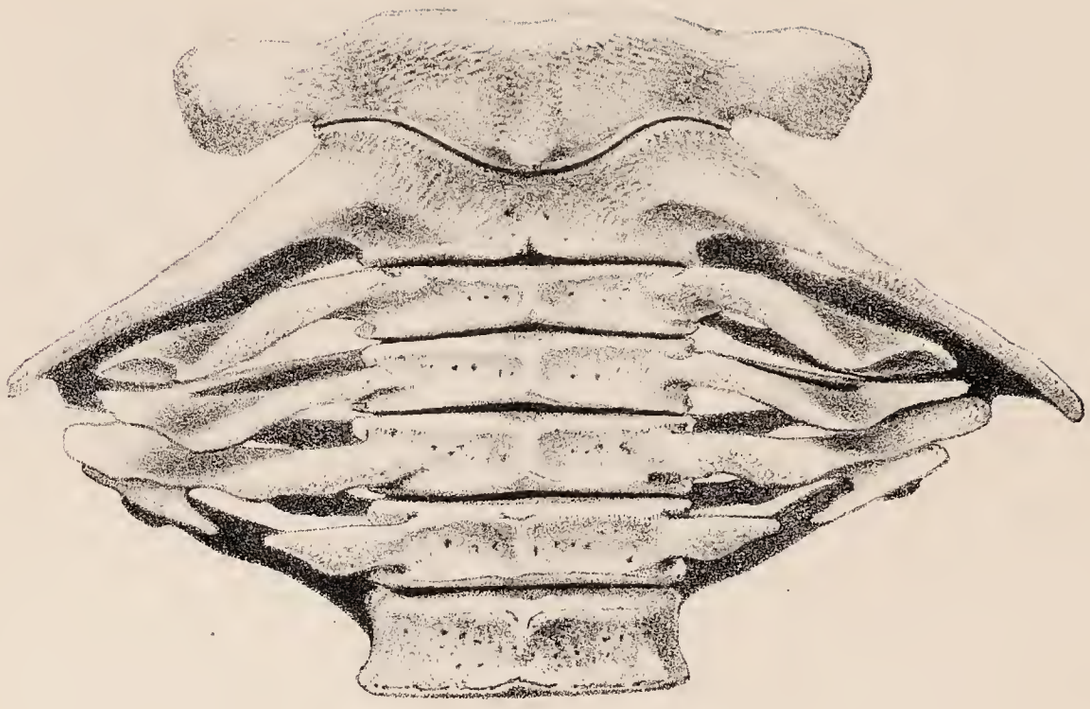

FIG. I.

Cervical Vertebrae in Balænoptera musculus, Peterhead, 1871. Under aspect. $\frac{1}{12}$.

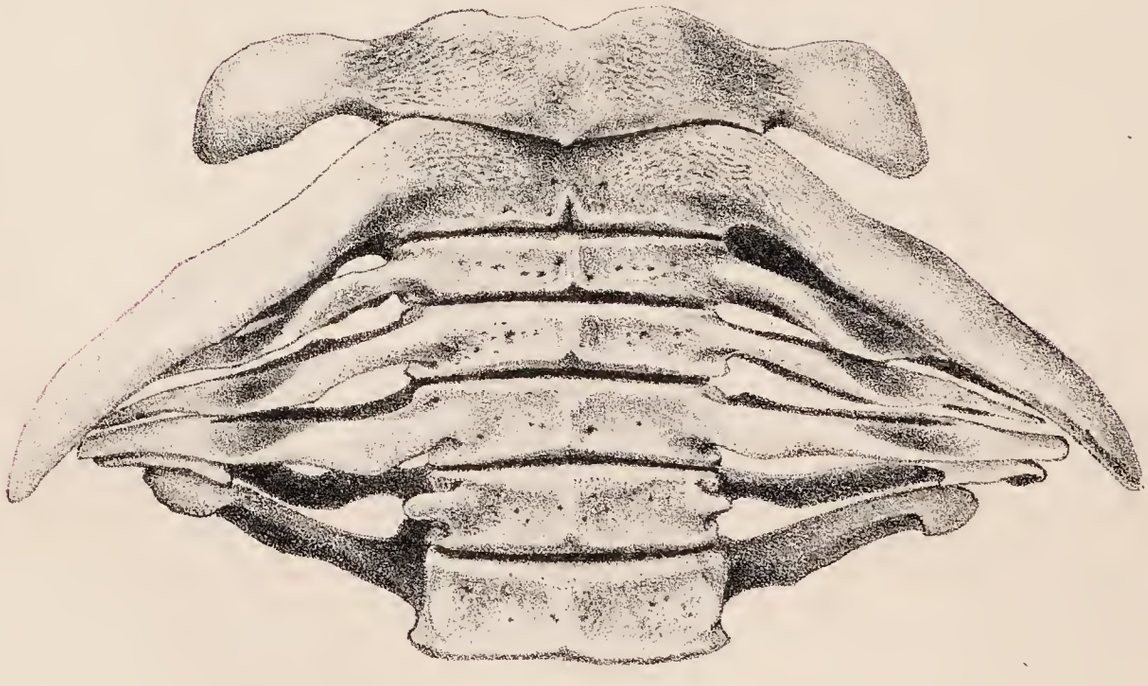

FIG. II.

B. musculus. Stornoway, 1871. Under aspect. $\frac{1}{12}$.

CERVICAL VERTEBRAE AND THEIR

Keith & Gibb Lith^rs Aberdeen.

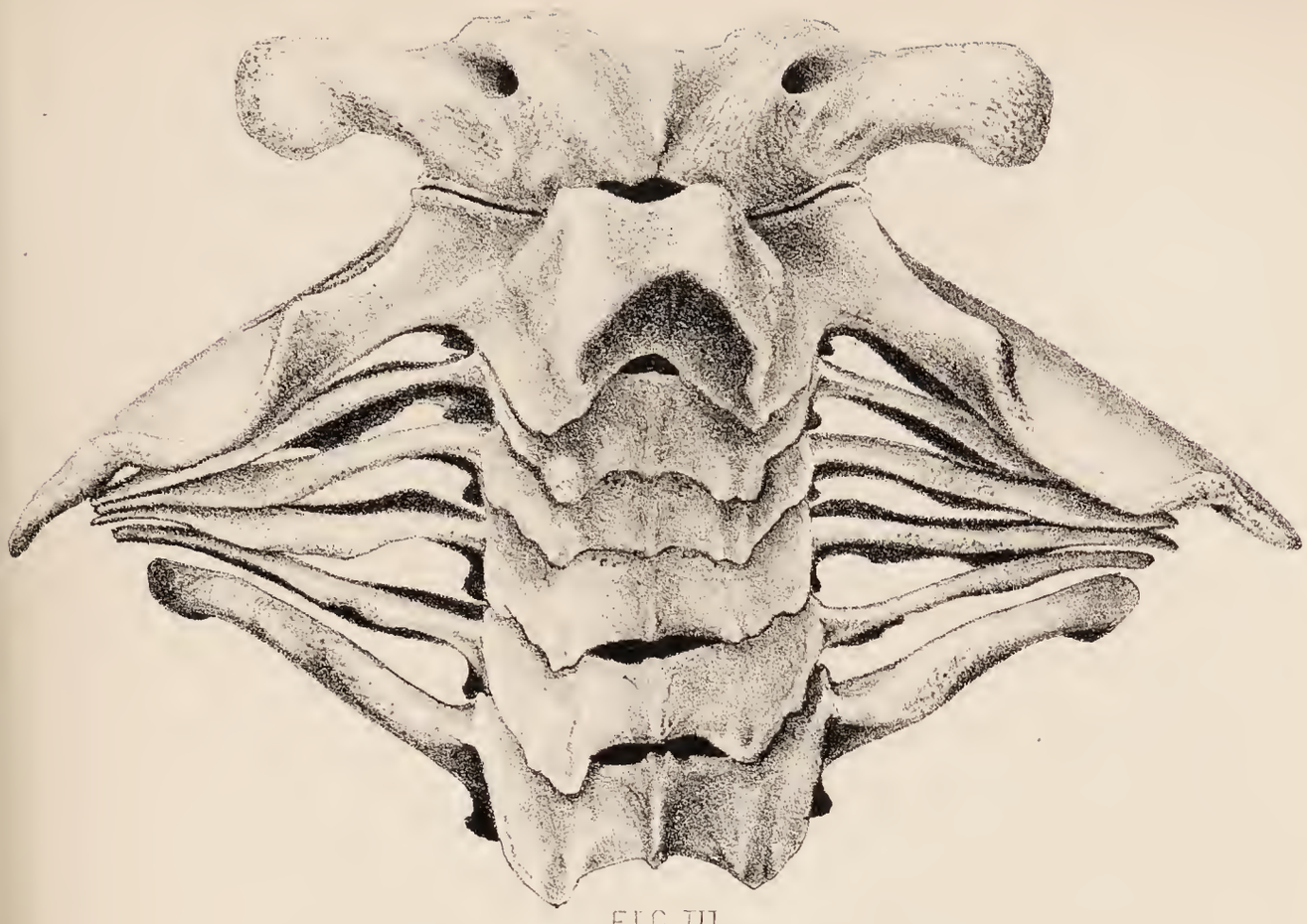

FIG. III.
B. musculus. Wick 1869. Upper aspect $\frac{1}{11}$

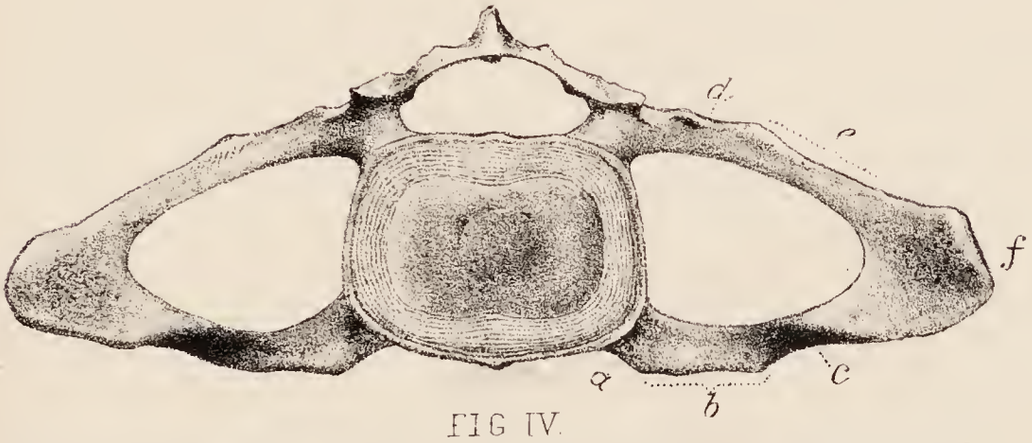

FIG IV.
Fifth cervical vertebra of B. musculus (Stornoway Fig 2) Front aspect $\frac{1}{12}$

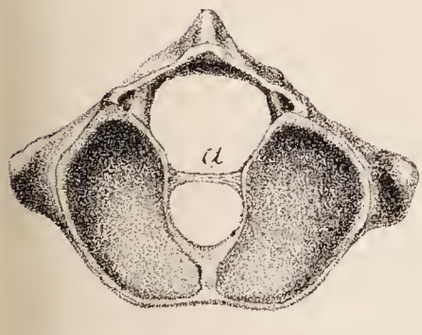

FIG. VI.
Atlas of young B. rostrata, with Transverse ligament. Aberdeen. 1870. Front aspect. $\frac{1}{6}$

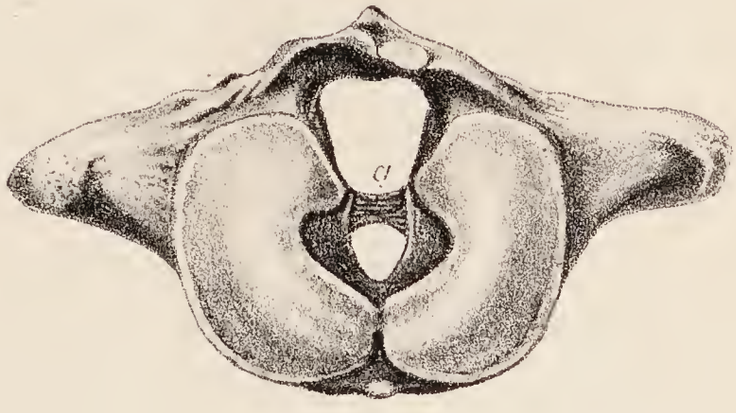

FIG. V.
Atlas of B. musculus with Transverse ligament (Wick Fig. 3) Hinder aspect $\frac{1}{12}$

ARTICULATIONS IN FIN-WHALES.